Pooling Data on Pools

Genotoxicity of Chemicals in Indoor Swimming Pools

Disinfection by-products (DBPs) form in swimming pool water from reactions between disinfectants such as chlorine and organic matter such as sweat, skin cells, and urine. A new study described in a set of three articles provides the first comprehensive characterization of DBPs in an indoor pool environment and offers initial evidence of cellular-level effects of these chemicals in swimmers in an indoor chlorinated pool [***EHP* 118(11):1523–1530; Richardson et al.**; ***EHP* 118(11):1531–1537, Kogevinas et al.**; ***EHP* 118(11):1538–1544, Font-Ribera et al.**].

Markers of genotoxicity and mutagenicity were detected in swimmers after 40 minutes in the pool.

The authors assessed short-term changes in 49 healthy adults after they swam for 40 minutes in a public indoor chlorinated pool. They observed increases in two biomarkers of genotoxicity relative to the concentration of brominated trihalomethanes (THMs) in exhaled breath, which were used as a proxy of the swimmers' total DBP exposures. Those biomarkers were micronuclei in blood lymphocytes (which have been associated with cancer risk in healthy subjects) and urine mutagenicity (a biomarker of exposure to genotoxic agents).

The team also took detailed measurements of THMs in air around the pool and in exhaled breath of the swimmers before and after swimming. The investigators measured several biomarkers of respiratory effects after swimming and found changes in only one—a slight increase in serum CC16, which suggests an increase in lung epithelium permeability. However, they found no evidence that DBP exposure affected lung function.

The research team identified more than 100 DBPs in the water of the chlorinated pool as well as another indoor pool disinfected with bromine. Some of these compounds had never been reported previously in swimming pool water and/or chlorinated drinking water. *In vitro* assays showed the swimming pool water was mutagenic at levels similar to that of drinking water but was more cytotoxic (could kill cells at a lower concentration) than drinking water.

The researchers acknowledge the need for further research on a variety of swimming pools under various conditions of maintenance and use as well as more complete evaluations of the uptake and potential effects of the compounds present in pool water. They also note the importance of timing in the collection of biological samples—a parameter for which there was no precedent, given the lack of previous studies of this type with swimmers. Above all, they emphasize that positive health effects of swimming can be maintained by minimizing pool levels of DBPs.

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Opening the Window to Cancer

Potential Mechanism behind Increased Susceptibility in Rats Exposed Prenatally to BPA

Exposure to environmental factors before birth or during other critical periods of development can cause subtle changes in a tissue's molecular foundations, leading to health effects later in life. Prenatal exposure to bisphenol A (BPA) is linked to cellular and structural changes in the mammary glands of adult rats and increased susceptibility to chemically induced cancer. New research now suggests a possible mechanism of action for this increase in cancer susceptibility via altered protein expression patterns in rat mammary gland tissue [***EHP* 118(11):1614–1619; Betancourt et al.**].

Pregnant rats were exposed to 0, 25, or 250 μg BPA/kg/d from day 10 to 21 postconception, and their female offspring were given a single dose of the cancer-inducing compound dimethylbenzanthracene (DMBA) at 50 or 100 days after birth (i.e., young adulthood). Experiments were conducted to determine the relationship between prenatal BPA exposure and the expression of proteins intrinsic to the growth and development of the mammary gland in adulthood. These proteins included estrogen receptor-α (ER-α); PR and Bcl-2, downstream targets of ER-α; steroid receptor coactivators (SRCs) 1 to 3, which influence ER-α transcriptional activity; and several growth factor receptors and signaling molecules that direct cell proliferation and programmed death.

Body weight and hormonally sensitive end points (time to vaginal opening, serum levels of the hormones 17β estradiol and progesterone, and estrous cyclicity) were assessed but found to be unchanged in relation to prenatal BPA exposure. ER-α, PR, and Bcl-2 were significantly downregulated and SRC-3 and some signaling molecules were upregulated in rats exposed prenatally to BPA.

DMBA administered to rats at 50 days did not yield significant differences in tumor incidence between those with or without prenatal BPA exposure. Among animals that received DMBA at 100 days, all assayed proteins were significantly upregulated in the BPA-exposed rats, and cell proliferation was enhanced, but apoptosis was unchanged. These animals also had decreased time to tumor formation and increased tumor incidence and severity.

These findings suggest that prenatal exposure to BPA in rats alters expression of key receptors and components of cellular signaling pathways in the mammary gland in adulthood, consequently increasing the tissue's susceptibility to chemically induced cancer. Whether this occurs in humans is unknown. Data from the Centers for Disease Control and Prevention indicate human exposure to the chemical is widespread, with approximately 95% of Americans estimated to have detectable levels of BPA metabolites in their urine. Continued research is necessary to elucidate the mechanisms by which BPA exposure early in life influences the development of permanent effects in maturity.

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